4:30

LOW ENERGY DIRECT CURRENT ABLATION OF RETROGRADE CONDUCTION IN PATIENTS WITH AV NODAL TACHYCARDIA

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Selective ablation of retrograde conduction was achieved in 9 pts with recurrent AV nodal tachycardia (AVNT) unresponsive to antiarrhythmic drugs. Low energy direct current (DC) ablation was performed using a 7F USCI bipolar contoured catheter and an ablator that delivers up to 40 joules and 3000 volts.
There were 2 males and 7 females, with a mean age of 58 (range 35-74) yrs. Three to 14 (mean 8.6±3.7) shocks were given during 1 or 2 sessions, with a mean cumulative energy of 233±111 joules, 18.8±10.7 kilovolts, 143±81 amps. An electrophysiologic study 2-5 days following ablation demonstrated VA block in 7 pts and retrograde Wenckebach at a pacing cycle length of 700 msec and 390 msec, respectively, in 2 pts. Anterograde conduction was modified in 6 pts: the AH interval increased by 109±38 msec, and dissociated anterograde AV nodal curves, present before ablation, were eliminated in all. In 2 pts, selective ablation of VA conduction was achieved without any effect on anterograde AV nodal properties. In 1 pt, who had failed previous high energy DC ablation, complete heart block was produced after shocks totalling 150 joules. This pt received a DDD pacemaker. During a mean follow-up of 7 months (range 1-17 months), 8 pts are asymptomatic without antiarrhythmic drugs. Only 1 pt had a recurrence of AVNT requiring medical treatment.
In conclusion, low energy DC ablation can selectively ablate retrograde conduction in pts with AVNT. In most cases, ablation also modifies anterograde conductio with elimination of dissociated AV nodal curves.

5:00

MODULATION OF AV NODAL CONDUCTION BY RADIOFREQUENCY CURRENT IN PATIENTS WITH AV NODAL REENTRANT TACHYCARDIA

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In 14 pts (1m, 13f, mean age 38 years) with AV nodal reentrant tachycardia (AVNRT, cycle length 331±40ms) 4 to 22 radiofrequency current (RFC, 300 kHz, 7-30 W, 10-60 s) applications (A) were delivered via electrode catheters (8 pts 6F 2 mm tip electrode, 6 pts 7F 4 mm) in unipolar mode to the AVN in order to suppress AVNRT by modulation of AVN conduction. In 12/14 pts AVNRT was no longer inducible after RFCA, permanent increase (≥20 ms) of AVN conduction time could be achieved (in antegrade direction in 11 pts, in retrograde direction in 10 pts with retrograde block (B) in 4/10 pts). Prolongation of AVN refractory periods could be achieved in 7/12 pts in antegrade (304±43 -> 381±55 ms) and in 6/12 pts in retrograde (317±52 -> 384±86ms) direction. In 1/12 pt AVB III° occurred 2 days after RFCA, the other 11 pts were free of recurrencies or complications during follow-up (13±9 months). In 1/14 pt no effect on AVN conduction could be achieved, in 1/14 pt permanent AVB III° did occur during RFCA. In the last 5 consecutive pts titration of RFC power was performed, beginning at each new catheter position with a low energy (10W/10s) and a gradual increase up to 35W/40s. In these pts AVB III° was never induced. Conclusions: 1) Modulation of AV nodal conduction was successful in 79 % of pts with drug refractory AVNRT. 2) In another 14% of pts AVB III° occurred during or after RFCA with consecutive pacemaker implantation and relief of symptoms. 3) RFCA proved to be safe without complications. 4) Careful energy titration may be necessary to avoid AVB III°.

4:45

CLINICAL EFFICACY OF AV NODAL MODULATION VERSUS ABLATION WITH RADIOFREQUENCY CURRENT IN ATRIAL FIBRILLATION/FLUTTER

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The aim of AV nodal (AVN) modulation with radiofrequency current (RFC) in pts with atrial fibrillation (Afib) is to control ventricular rate without antiarrhytmic drugs(AA) and maintain 1:1 conduction (C) during SR, thereby avoiding pacemaker (PM) implantation. 40 pts with drug refractory (6±2 AA) Afib (33), flutter (5) or ectopic tachycardia (2) were treated by 10±7 applications of 328±193 J betweeen the tip electrode of the catheter at the proximal AVN and a back plate until prolongation of AVNC (AH increase >50%, WCL>400ms) or AVB III° occured. In 9 pts a lasting effect coud not be achieved by RFC, and additional 1-4 DC shocks were subsequently applied via the same catheter. At the end of the ablation procedure AVB III° was present in 23 pts (group A), AVN modulation (AH 79±26->147±55, WCL 351±77->455±108ms) in 16 pts (group B). During a follow up of 16±12 months resumption of AVNC occured in 6/23 group A pts leading to recurrent symtoms, controlled with previously ineffective AA in 5/6. Permanent (1) or intermittent (6) AVB III° developed after the procedure in 7/16 group B pts leading toPM implanatation. Symptoms were controlled in 6/7. Out of the other 9 Group B pts 4 (10% of all) became asymptomatic, 1 improved with AA and 4 underwent a 2nd ablation procedure

Control of symptoms:	Group A(n=23)	Group B(n=16)
-without AA	78%	63%
-with AA	+17%	+ 6%
PM implantation	87%	44%

Conclusions: 1)Lasting and clinically effective prolongation of AVNC without subsequent need for PM implantation or antiarrhythmic therapy could only be achieved in 10% of pts with atrial fibrillation/flutter. 2)AVN ablation by RFC is an efective treatment for these pts, but requires PM implantation. 3)AVN modulation can not be recommended because of its low clinical efficacy.

5:30

RETROGRADE AV NODAL CONDUCTION CAN BE SELECTIVELY ELIMINATED BY NEODYMIUM:YAG LASER PHOTOCOAGULATION IN DOGS

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AV nodal reentry requires intact retrograde (VA) conduction. The purpose of this study was to test the feasibility of selective elimination of VA conduction by limited laser photocoagulation (LPC) of the AV node, and to analyze the histologic substrate of unidirectional VA block. Nd:YAG LPC was performed during cardiopulmonary bypass through a right atriotomy in 15 dogs that had intact VA conduction prior to surgery. Short laser pulses (20 W, 0.5 cm spot size) were delivered to an area between the coronary sinus orifice and the site of the most proximally recorded His deflection. End point of LPC was acute development of second degree AV nodal block at a paced atrial cycle length of 250 ms. 11/15 animals (group I) acutely developed complete VA block, while AV conduction persisted in all. In 4/15 animals (group II), VA conduction remained intact. During a 3-month follow-up, VA conduction remained absent in all group I animals. VA block was not reversed by isoproterenol infusion. Anterograde AV nodal characteristics (Wenckebach cycle length, functional refractory period, ventricular rate during atrial fibrillation) were unchanged in 5 and modified in 6. Complete AV block was not observed. In 4 control cases (sham operation), AV and VA characteristics stayed unchanged. Histology at 3 months in cases with unidirectional VA block showed the compact part of the AV node intact with destruction of the atrial approaches and the superficial layers of the proximal end of the node on the right side. Conclusions: (1) VA conduction is more sensitive to LPC than AV conduction. (2) Destruction of the atrial approaches on the right side with preservation of the compact part of the AV node is probably required for selective elimination of VA conduction. (3) Graded Nd:YAG LPC of the AV node may become a promising new technique for the treatment of AV nodal reentrant tachycardias.